CASE REPORT

Feline Urological Syndrome

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Summary

A case of feline urolithiasis is described. It was demonstrated that serial determinations of renal function were mandatory in order to provide a favorable prognosis. Because hyperkalemia was present, the fluid of choice for the initial treatment was saline. The various considerations in the prevention, treatment and monitoring of cats with feline urolithiasis syndrome are discussed.

Résumé

Un cas du syndrome urologique du chat

L'auteur décrit un cas d'urolithiase féline et démontre la nécessité d'une série de déterminations de la fonction rénale, afin d'obtenir un pronostic favorable. La présence d'une hyperkaliémie faisait de la saline le traitement initial de choix. L'auteur commente les diverses considérations relatives à la prévention, au traitement et à la surveillance de cette condition.

Introduction

Although the feline urological syndrome (FUS) is not a new topic in the veterinary literature, the etiopathogenesis (3, 4, 5, 6, 7, 8, 9, 10) has not been established. Factors which have been investigated include viruses (3, 4), diet (6, 7, 8, 9), struvite (11), narrowed penile urethra (10) and bacterial infection of the urinary tract (11). Reports have indicated that age, weight, level of activity and diet were all associated with the disease and that between 0.6% and 1% of the total domestic cat population was affected (14, 15).

History

A five year old, castrated male, Sealpoint Siamese cat, was presented to the Western College of Veterinary Medicine in a severely depressed state. The owner stated that the cat had been lost for 48 hours.

Clinical Signs, Laboratory Data and Treatment

The cat was recumbent, nonresponsive to stimuli, and severely dehydrated (± 10%) as determined by skin turgor. The rectal temperature was 34.8°C. The femoral pulse rate was 132 beats per minute and very weak. Capillaries of the oral mucous membranes had a slow refill time. Heart sounds were of low intensity and no murmurs or arrhythmias could be heard. A large, hard, movable mass was found on palpation of the abdomen. The penis extruded from the prepuce and a small white plug appeared to be obstructing the urethral opening.

The cat was admitted to the hospital with a diagnosis of urethral obstruction. The obstruction was relieved by massage of the penis and flushing the urethra with saline through a flexible catheter.[1] The catheter was then inserted into the bladder and sutured to the prepuce. Severe hematuria and crystalluria were present. Urine was collected for urinalysis, quantitative culture and antimicrobial sensitivity. The bladder was emptied.

The bladder was flushed with a chilled solution of saline and furacin. An Elizabethan collar was put around the animal's neck and a catheter was placed in the jugular vein. Blood was taken for complete blood count (CBC), blood urea nitrogen (BUN), serum creatinine and electrolytes (Table I and II). Although electrocardiogram tracings characteristic of hyperkalemia were not observed, it was assumed that the cat was hyperkalemic and acidotic. Intravenous fluid (0.9% saline) containing no potassium was chosen for hydration therapy. Sodium bicarbonate, 10 mEq was added to each 250 ml of saline. The fluid was warmed to prevent a further increase in the hypothermia. Antibiotic therapy, 100 mg Ampicillin[2] four times daily (q.i.d.) intravenously, was instituted and the cat was put in a heated cage and monitored.

The rectal temperature dropped initially (34°C) but after four hours it started to rise (35°C). The next morning the rectal temperature had risen to 38.3°C. Blood was again collected for CBC, BUN, serum creatinine and electrolytes. As the serum potassium level had dropped from 7.3 mEq/L to 5.2 mEq/L, fluid therapy was changed to Ringer's solution containing 4 mEq/L potassium to prevent hypokalemia from developing as a result of a potential postobstructive diuresis. An encouraging sign was the dramatic drop in creatinine and BUN from 18.9 mg/dl and 280 mg/dl to 4.3 mg/dl and 82 mg/dl respectively. Although the cat had begun to take fluids orally, IV fluids (Ringer's) were continued for an additional two days after which

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[1]Open end tomcat catheter 3½ Fr., Sherwood Medical Industries, Inc., St. Louis, Missouri 63103.
[2]Ampicillin, Ayerst Laboratories, Montreal, Quebec.

TABLE I

HEMATOLOGICAL VALUES OF PRESENT CASE

	Normal Values for WCVM Laboratory	Day of Admission	Day 1	Day 3	Day 5
WBC x 10^3/mm^3	5.5- 19.5	13.8	13.3	22.0	15.8
RBC x 10^6/mm^3	5.0- 10.0	8.6	5.9	5.6	5.44
HGB/gm	8 - 15	15.8	10.1	8.3	8.8
PCV %	24 - 45	43.8	31.1	25.1	26.1
MCV μ^3	39 - 55	51	53	50	48
MCH $\mu\mu$g	13 - 17	18.7	17.3	16.7	16.4
MCHC %	30 - 36	36.7	32.7	33.2	33.6
Neutrophils	2500 -12500	12006	7847	16940	12956
Bands	0 - 300	1242	4389	660	158
Lymphocytes	1500 - 7000	414	931	3520	1896
Eosinophils	0 - 1500	—	—	440	474
Monocytes	0 - 850	—	—	440	158
Reticulocytes%	0 - 1.0	1.0		—	—
Thrombocytes x 10^5/mm^3	3.0- 7.0	Normal	Low	Normal	Normal

subcutaneous fluids were administered for an additional two days. The urinary catheter was removed on day 3. *Escherichia coli* was cultured from the initial urine specimen. As it was sensitive to Ampicillin, this antibiotic was continued for 14 days. During the cat's hospitalization the urine pH fluctuated between 6-6.5. Urine acidifiers were never used.

Discussion

The feline urological syndrome is a combination of entities including cystitis, urethritis and urolithiasis. The syndrome in male cats is frequently associated with feline urethral obstruction (FUO).

Although the virus-induced etiology has been questioned (8), Fabricant (2, 3) felt that she had proof for virus-induced urolithiasis. She hypothesized that the virus caused proliferation of other latent virus(es) in the bladder which in turn set off a mechanism resulting in urolithiasis. However, no additional support for the viral theory has appeared in the literature.

Dietary influences have been widely investigated (1, 4, 10). In one of these studies (10) it was shown that an increase in the content of magnesium and phosphate in the diet resulted in urethral obstruction. Similarly, the addition of calcium (1) to 2% of the diet increased calculi formation. Although the addition of 1% methionine did not prevent the formation of uroliths, it did prolong the time required for obstruction to occur. It would appear from these studies that calculi formation was dependent on the calcium to phosphorus to magnesium ratio in the diet. In addition, 15% alanine in the diet also prevented the formation of calculi (1).

Struvite crystals composed of ammonium, magnesium and phosphate apparently play an important role in the etiopathogenesis of urethral obstruction. Rich (11) reported that struvite crystals were found in both affected and unaffected cats. He noted that there is a direct correlation between increasing pH and crystal precipitation. Acid urine, below pH 6-6.5 apparently caused many crystals to dissolve.

Although a narrow penile urethra has been incriminated as a cause of urolithiasis, Herron (7) showed that early castration did not affect the urethral circumference. Urinary tract infections are rarely the primary cause of urethral obstruction (1). Although less than 50% of urolithiasis cases had bacteriuria it was suggested that bac-

TABLE II

BLOOD CHEMISTRY VALUES OF PRESENT CASE

	Normal Values for WCVM Laboratory	Day of Admission	Day 1	Day 2	Day 3	Day 4
Sodium (mEq/L)	150-156	147	162	160	157	157
Potassium (mEq/L)	4.0 - 5.8	7.3	5.2	4.0	4.3	4.0
Chloride (mEq/L)	117-123	92	126	123	120	126
Calcium (mg/dl)	7- 9	8.8				
Phosphorus (mg/dl)	5.3 - 7.6	18.4	7.9	5.6		
BUN (mg/dl)	20- 30	279.6	81.6	37.5	25	17.5
Creatinine (mg/dl)	1.0 - 2.0	18.9	4.3	2.5		1.3
Total protein (gm %)	5.9 - 7.9	9.6	7.1	—	5.6	6.8

teriuria could influence the recurrence of urolithiasis (11). It is likely that FUS may be associated with an interaction of the above mentioned factors and perhaps with factors as yet undetermined.

It would appear that in the present case the obstruction caused severe dehydration from a lack of water intake, insensible loss and perhaps vomition (not noticed by the owner). The tremendous increase in BUN and creatinine values (Table II) probably resulted from postrenal uremia. A single determination of BUN and creatinine concentrations, regardless of the values obtained, does not provide a reliable indication of the reversibility or irreversibility of the underlying renal damage (9). In the present case, the BUN and creatinine values dropped dramatically within 12-24 hours providing a reasonable prognosis. Tissue catabolism results in the movement of potassium from the intracellular space to the extracellular space in exchange with hydrogen ion. Therefore, hyperkalemia is common in cases of acidosis (6). Although not evidenced in the present case, hyponatremia may result from vomition, anorexia, sequestration in the bladder and the movement of sodium intracellularly in exchange for potassium (7). Hyperphosphatemia was probably a consequence of anuria (6).

The initial and most important treatment in handling a case of FUS is to establish a patent urethra. Although the animal was so depressed that manipulation of the penis did not cause any degree of discomfort, a topical anesthetic may be used. If restraint had been necessary, an ultra shortacting barbiturate could have been given. Gas anesthesia can also be used in severely affected animals. However, drugs that are metabolized primarily by the kidneys should be avoided.

Once urethral patency has been obtained, a tomcat catheter should be sutured into place to monitor urine output. In addition, it seems beneficial to rinse the bladder with saline (through the catheter). A chilled solution of saline and furacin may be used to stop hemorrhage in cases of severe hematuria. Parenteral fluid therapy should be initiated as soon as possible. Saline is the fluid of choice in the initial treatment. It contains more sodium than Ringer's and Lactated Ringer's and does not contain potassium. Hyponatremia and life-threatening hyperkalemia are nearly always present in FUS. An electrocardiogram (ECG) may provide a clue as to serum electrolytes (12).

Hyperkalemia can also be reversed by the administration of regular insulin IV. This causes potassium to be driven intracellularly. If hyperglycemia is not present at the time of insulin administration, glucose may have to be administered concurrently (12). Others have recommended that the hyperkalemia may be reversed by administration of a multiple electrolyte solution (6) after the urethral obstruction has been corrected.

The amount of fluid (ml) necessary to rehydrate the animal may be calculated as follows:

$$\%\ \text{dehydrated} \times \text{body weight (kg)} \times 10$$

For example, a 4 kg cat that is 10% dehydrated would require $10 \times 4 \times 10 = 400$ ml of replacement fluid. Maintenance requirements are roughly 50 ml per kg per day or about 2 ml/kg/hour.

Acidosis may be combatted by the addition of sodium bicarbonate to the saline. The base deficit was estimated to be 15 mEq of bicarbonate. The total milliequivalents of bicarbonate needed to correct the acidosis for 24 hours may be obtained by multiplying base deficit x 0.5 x body weight (kg). Twenty five percent of the total dosage was given IV and the remainder was added to the IV fluids.

A period of diuresis may follow the relief of urinary tract obstruction (13). This diuresis may cause hypokalemia and other alterations in serum electrolytes. If hypokalemia does develop, potassium should be supplemented accordingly (13). The diuretic phase is important in the sense that there is a tremendous output of urine and rehydration with IV fluids during this period must take place. In the present case the diuretic phase lasted three to four days. After that period fluid therapy was gradually decreased.

Another consideration in treatment is the urine pH. If the urine is alkaline, acidifers should be administered in order to decrease the pH to at least 6.5. However, one should not start to acidify the urine before the animal's acidotic state has been corrected. Urine alkalinity was not a problem in this case. Salt (NaCl) tablets have been recommended to increase the water consumption and to cause a dilution effect of the urine. However, salt tablets may cause vomition in an anorexic cat. Therefore, enteric coated tablets are preferred.

Antibiotics are also indicated in the treatment of FUS. Although bacteriuria has been recorded in less than 50% of cases, antibiotics are of benefit in the placement of jugular and urinary catheters. Antibiotics of choice should be nonnephrotoxic drugs and excreted by the kidney. In this case ampicillin was used for two weeks.

The prevention of recurrence of FUS may be attempted by diluting the urine, lowering magnesium content of urine by feeding low magnesium diets and lowering urine pH where applicable.

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LETTERS TO THE EDITOR

Cutaneous Melanoma in Hampshire Swine

DEAR SIR:

There are references in recent literature (as well as some unpublished material) to the incidence of cutaneous melanoma in Duroc swine. The disease is characterized by raised, black areas on the skin which may break and drain. In some cases, it will become systemic and result in condemnation of the carcass at slaughter. The condition seems to have some genetic relationship and therefore is of economic importance to the conscientious breeder selling breeding stock.

We have observed and confirmed a case of cutaneous melanoma in a Hampshire boar. What appears to be cutaneous melanoma was also observed, but not confirmed, in a Hampshire sow. The black pigmented areas of the sow began to turn white soon after the suspect lesions appeared.

If other practitioners are observing this type of lesion in Hampshire animals, with or without a change of skin pigmentation, we would ask that they have the diagnosis confirmed histologically and contact the undersigned.

Yours truly,

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Letters to the Editor to Contain Practice Information

DEAR SIR:

Since my graduation in 1966, I have been somewhat disappointed in the lack of written practical material for small animal practitioners. Last year I wrote a long and confidential letter to the editorial board making many suggestions as to how I felt the journal could be improved. To my pleasure they decided to act on some of the suggestions, and to my horror they asked me to serve as an assistant editor.

My main aim will be to work on the establishment of a "practitioners forum" so that people making minor discoveries in practice can share experiences in the journal. Many of the little tips which make practice so much easier were learned in the coffee shops and bars at professional meetings or were disseminated by the more helpful drug company representatives.

For the time being it has been decided to put this information in the "letters to the editor" section. While the material will be edited the format and references may vary more than the research papers do. It is hoped that many more practitioners will send information to the journal so we can find out what is really happening in applied veterinary medicine in Canada.

Yours truly,

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